Amendments to the Specification

Please amend the specification at paragraphs [0040], [0041], [0043], [0046], [0047] and [0048], as follows:

[0040] FIG. 2 demonstrates that the recognition of HA by NKp46 is mediated through the membrane proximal domain (NKp46D2). Uninfected and SV-infected 721.221 cells were incubated with or without monoclonal antibody (mAb) against HA (135.7) or control mAbs (12E7 and TC-9A1) and stained with the fusion proteins NKp30-Ig (Fig. 2a), NKp46-Ig (Fig. 2b), NKp46D2-Ig (Fig. 2c), or NKp46D1-Ig (Fig. 2d), followed by PE-conjugated goat antihuman antibodies.

[0041] FIG. 3 demonstrates that the binding of the membrane proximal domain to viral HA is sialic acid dependent. NKp46D1-Ig (Figs. 3b and 3c) or NKp46D2-Ig (Figs. 3a and 3d) cells were treated with NA (filled bars) or with PBS as control (empty bars). SV- (Figs. 3a and 3b) or IV- (Figs. 3c and 3d) infected and uninfected 721.221 or 1106mel cells, respectively, were washed and stained with either the PBS-treated or NA-treated fusion proteins followed by PE-conjugated goat antihuman Fc antibodies.

[0043] FIG. 5 demonstrates that a specific mAb directed against the membrane distal domain (D1) of NKp46 do not block NK-mediated lysis of target cells. NK cells (Fig. 5a), BW (Fig. 5b) and BW transfected with NKp46 (Fig. 5c) were incubated with and without 461-G1 (thick black line) or control 12E7 (thin black line). Fig. 5d shows the results of ELISA, in which the indicated immobilized fusion proteins were incubated with 461-G1 or no antibody, followed by incubation with HRP-conjugated rabbit antimouse IgG as secondary mAb. Fig. 5e shows the results of SDS-PAGE and Western blot analysis of the fusion proteins NKp30-Ig (lane 1); NKp46D2-Ig (lane 2); NKp46D1-Ig (lane 4); NKp46-Ig (lane 5); KIR2DL1-Ig (lane 6), and the control low-protein medium (lane 3). The marker is indicated by M. Fusion proteins were analyzed with mAb 461-G1 and HRP-conjugated rabbit antimouse IgG. Fig. 5f shows the results of ^{35}S-labeled cells incubated with no antibody (□), anti-CD16 mAb (B73.1.1; ♦), anti-NKp46 mAb (461-G1; ○) or anti-CD99 mAb (12E7;▲), followed by addition of NK cells in effector to target ratio (E/T) of

3:1. Fig. 5g shows the results of pre-incubating NK cells with the indicated mAbs, followed by incubation with either uninfected 1106mel cells or 1106mel cells infected with the indicated influenza strains, at E/T of 20:1.

[0046] FIG. 8 demonstrates that amino acid residue Thr225 of NKp46 is crucial for the recognition of tumor cells by NKp46. 1106mel cells were stained with the wild-type NKp46-Ig (Fig. 8a) or with the sugar-mutated forms NKp46T125A (Fig. 8b), NKp46N216A (Fig. 8c), NKp46T225A (Fig. 8d), or NKp46T225N (Fig. 8e), followed by PE-conjugated goat antihuman Fc.

[0047] FIG. 9 Titration of NKp44-Ig, NKp44D-Ig and NKp44LP-Ig binding to tumor cells. PC-3 cells (Fig. 9a) or HeLa cells (Fig. 9b) were incubated with the indicated fusion proteins, followed by washing and incubation with FITC-conjugated goat antihuman Fc secondary antibody.

[0048] FIG. 10 demonstrates the binding of NKp44-Ig, NKp44D-Ig and NKp44LP-Ig to viral-infected cells. Uninfected 1106mel cells (thick black line) and 1106mel cellsinfected with IV (thin black line) were incubated with fusion proteins NKp44-Ig (Fig. 10a), NKp44D-Ig (Fig. 10b), NKp44LP-Ig (Fig. 10c), or with secondary antibody as control (Fig. 10d), followed by staining with FITC-anti-Fc secondary antibody.